Editorial

Beyond Spirals

Ventricular fibrillation is the major cause of sudden cardiac death, and spiral- or vortex-type reentry rotating around functional obstacles (rotors) is now considered to be the central organization machinery of VF. The rotors, like typhoons or hurricanes, create turbulent areas of ventricular excitation to compromise mechanical performance. Regulation of rotors by pharmacological and non-pharmacological procedures is, therefore, a central issue to be addressed for the efficient prevention of sudden arrhythmic death. Two major VF mechanisms have been proposed in terms of rotor dynamics: dynamic wavebreak and mother rotor. The former postulates that VF results from the instability of rotors, which ultimately leads to continuous breakup. Dynamic factors such as restitution properties in association with structural and/or functional heterogeneity are supposed to have synergetic effects to increase the chance of continuous breakup. The latter hypothesizes that VF is maintained by a wavefront emanating at an exceedingly high frequency from a relatively stable rotor (driver) anchored (or pinned) at certain morphological discontinuities, and the multiple wavelets that characterize VF are epiphenomena caused by fibrillatory conduction block in the periphery of the rotor. These two mechanisms are not totally incompatible, but may contribute in a complementary manner in various heart diseases. Recently, we demonstrated, by using a high-resolution optical action potential mapping system in rabbit hearts, that pharmacological blockage of the rapid activation component of delayed rectifier K^+ current (I_{Kr}) or moderate lowering of temperature (hypothermia) facilitates early spontaneous termination of VT/VF resulting from spiral-type reentry (Am Heart J 2007, 2008). These procedures were shown to cause considerable drift of rotors, and their subsequent annihilation via collision. Blockade of inward rectifier K^+ current (I_{K1}) was reported by other investigators to have similar effects on rotor dynamics in favor of early VF termination. Based on these observations, blockade of I_{Kr} (or I_{K1}) or moderate hypothermia is expected to be effective in preventing VF, if the VF is attributable to rapidly circulating mother rotors, as they destabilize rotation activity. Failure of ventricular defibrillation by DC shock is mainly attributable to induction of new mother rotor-type excitations, giving rise to reinitiation of VF. I_{Kr} (or I_{K1}) blockade and moderate hypothermia could be effective in preventing such shock-induced reinitiation of VF via inhibition of anchoring or pinning of the rotors. However, these procedures have a potential risk to increase VF perpetuation, if it is due to continuous breakup of wave fronts. They may work like a double-edged sword. As a corollary, there is still much work required to achieve the development of ideal strategies beyond spirals for the treatment of serious recurrent VF and for the prevention of sudden arrhythmic death.

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